Clinical Assessment and Management of Portal Hypertension

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Abstract

The development of portal hypertension in a patient with cirrhosis portends a poor prognosis. Untreated or progressive portal hypertension has serious clinical outcomes, which are often fatal. It is important to recognize portal hypertension early to delay progression and to treat complications of portal hypertension as they arise. This review will focus on the clinical assessment and management of portal hypertension.

Keywords

- portal hypertension
- hepatic venous pressure gradient
- esophageal varices
- ascites
- encephalopathy

Objectives: Upon completion of this article, the reader will be able to (1) identify different approaches for noninvasive assessment of portal hypertension; (2) assess the complications of portal hypertension and their management; (3) discuss the prognosis associated with different manifestations of portal hypertension.

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Portal hypertension (PH) is a frequently encountered clinical syndrome due to a pathological increase in the venous pressure within the portal system. Hemodynamically, PH is defined by an increase in the venous pressure gradient across the liver, calculated from its inflow through the portal vein versus its outflow through the hepatic veins, with resultant value above normal (1–5 mm Hg).[1] The best method to definitively assess portal pressure is through catheterization of the hepatic vein with determination of the hepatic venous pressure gradient (HVPG), which is the difference between the wedged hepatic venous pressure (WHVP) and the free hepatic venous pressure (FHVP).[2]

In the Western world, cirrhosis is by far the most common cause of PH, accounting for 90% of cases.[3] In cirrhosis, PH is primarily a consequence of increased intrahepatic resistance to portal flow from a combination of functional abnormalities. These abnormalities lead to endothelial dysfunction, subsequent insufficient release of vasodilators, and increased hepatic vascular tone from structural disturbances associated with advanced liver disease. The structural disturbances of cirrhosis include increased fibrous tissue, vascular distortion from regenerative nodules, and microthrombi, all of which account for ~70% of total hepatic vascular resistance.[4] Splanchnic vasodilation, an adaptive response to changes in these intrahepatic hemodynamics, results in an increase in portal venous inflow as well and further aggravates the increase in portal pressures and, together with PH, contributes to the major sequela observed clinically.

Noncirrhotic Portal Hypertension

While cirrhosis is the most common cause of PH, any condition that interferes with blood flow within the portal system can

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conceivably cause PH. PH in the absence of cirrhosis, a condition referred to as noncirrhotic portal hypertension (NCPH), can be classified based on the site of resistance to blood flow as prehepatic, intrahepatic, and posthepatic.[3]

Prehepatic PH is often caused by portal vein thrombosis (PVT), with 70% of cases due to hypercoagulable syndromes in adults (myeloproliferative disorders such as polycythemia vera and essential thrombocytosis, factor V Leiden mutation, protein C or S deficiency, antiphospholipid syndrome).[5] As the insult occurs proximal to the liver, the WHVP, an indirect measure of sinusoidal pressures within the liver, will be normal and give rise to a normal HVPG.[2]

Intrahepatic PH can be further subdivided, in most cases, according to the results of hepatic vein catheterization.[5] Presinusoidal PH gives rise to normal or slightly elevated WHVP and FHVP measurements with subsequent normal HPVG values, as is the case for idiopathic PH/noncirrhotic portal fibrosis, early stages of primary biliary cirrhosis, and granulomatous diseases, such as schistosomiasis, sarcoidosis, and tuberculosis.[6] Sinusoidal PH gives rise to elevated WHVP and normal FHVP measurements with resultant elevated HVPG values, as is the case for most chronic liver diseases.[5] Postsinusoidal PH gives rise to elevated WHVP and normal FHVP measurements with resultant elevated HVPG values, as is the case for sinusoidal obstruction syndrome and Budd–Chiari.[2]

Posthepatic PH is typically caused by right heart failure (as in constrictive pericarditis and tricuspid regurgitation), which gives rise to elevated WHVP and FHVP measurements, resulting in a normal/near-normal HVPG.[5]

Knowledge regarding the natural history and clinical outcomes of NCPH is based on a limited number of studies, with current recommendations for the management of patients with NCPH similar to those for cirrhosis. In a recent study comparing the clinical presentation and outcomes of patients with NCPH versus compensated cirrhosis, the rate of progression to varices at risk of bleeding and the incidence of first variceal bleed were significantly higher in patients with NCPH, suggesting that the management of patients with NCPH (in particular, the timing of surveillance endoscopy for esophageal varices [EV]) should take into consideration the natural history of PH in these patients and not be simply derived from the observation of cirrhotic patients.[7] With the paucity of data on this subject, more studies are needed to further guide management of patients with NCPH.

Clinically Significant Portal Hypertension

As stated previously, PH from advanced chronic liver disease can be defined hemodynamically by an HVPG above 5 mm Hg. Based on portal pressures, patients can be further divided into those with mild or subclinical PH (HVPG $>$ 5 but $<$ 10 mm Hg) and those with clinically significant portal hypertension (CSPH), defined by an HVPG $\geq$ 10 mm Hg. Above this critical threshold of 10 mm Hg, patients with CSPH are at an increased risk of developing gastroesophageal varices, overt clinical decompensation (ascites, variceal hemorrhage [VH], and hepatic encephalopathy [HE]), postsurgical decompensation, and hepatocellular carcinoma.[2] In patients with compensated cirrhosis and PH, but without gastroesophageal varices, an HVPG of less than 10 mm Hg has a 90% probability of not progressing to clinical decompensation over a 4-year period.[8] Furthermore, each 1 mm Hg increase in HVPG is associated with an 11% higher risk of clinical decompensation; that is, a patient with a baseline HVPG of 15 mm Hg has a 55% higher chance of decompensating compared with a patient with an HVPG of 10 mm Hg, at equivalent model for end-stage liver disease (MELD) and albumin levels.[8] Severe PH (HVPG $\geq$ 12 mm Hg) and very severe PH (HVPG $\geq$ 16 mm Hg) are associated with an increased risk of variceal bleed and an increased mortality risk, respectively.[9] CSPH significantly increases the risk of 3- and 5-year mortality and of clinical decompensation after surgery for hepatocellular carcinoma.[10] In contrast, longitudinal studies have demonstrated that if the HVPG falls to less than 12 mm Hg or decreases by at least 20% from baseline values, either with drug therapy or spontaneously, then variceal bleeding may be prevented.[5,11]

Given the significant risk of clinical decompensation associated with CSPH, all patients with cirrhosis should be screened for the presence of CSPH at disease onset.[2] The appearance of new abdominal portosystemic collaterals and spleen enlargement during serial imaging is strongly associated with variceal formation/progression and CSPH, respectively.[12] Therefore, when performing screening for hepatocellular carcinoma, imaging evidence of worsening PH should be specifically sought.[2] Per the most recent Baveno VI conference in 2015, imaging showing collateral circulation is sufficient to rule in CSPH in patients with compensated cirrhosis of all etiologies.[13]

Clinical decompensation marks the symptomatic phase of cirrhosis, which is associated with a much higher mortality rate. At this stage, 100% of patients have CSPH.[2] As such, assessment of CSPH is relegated to patients with asymptomatic chronic liver disease or "compensated" cirrhosis in an effort to risk-stratify such patients.

Noninvasive Assessment of Clinically Significant Portal Hypertension

Physical exam findings that most specifically correlate with CSPH include spider nevi[14] and visible abdominal portosystemic collaterals; however, their absence cannot be used to rule out CSPH. Ultrasonography (US) is the first-line imaging technique for diagnosis and follow-up of patients with PH because of its noninvasiveness, cost-effectiveness, and ability to be performed at bedside. The presence of portosystemic collaterals (patent paraumbilical vein, splenorenal collaterals, dilated left- and short-gastric veins) in combination with inversion of flow within the portal system is 100% specific for CSPH.[6] In patients with compensated cirrhosis, US-Doppler has $>$80% specificity in diagnosing CSPH; however, sensitivity only approaches 40 to 70%.[6] Therefore, absence of specific ultrasound findings cannot rule out CSPH (►**Table 1**).

Noninvasive serum markers, alone or in combination, have been evaluated for their predictive value in PH, with low platelet count being the most common laboratory sign of PH. The degree of thrombocytopenia does correlate slightly with increased HVPG and the presence of gastroesophageal

Table 1 Comparison of the sensitivity and specificity (expressed as a percentage) of noninvasive tests in predicting clinically significant portal hypertension (CSPH) and high-risk esophageal varices (EV)

	CSPH		High-risk EV	
Noninvasive test (reference study)	Sensitivity	Specificity	Sensitivity	Specificity
US[6]	40–70	>80		
Platelets[20]			80	68
APRI[18]	66	73		
FibroTest[19]	88	50		
Platelet-to-spleen ratio[20]			85	66
LS[9]	87	85	86	59
SS[27]	98	74	83	57
LSPS[30,31]	83	84	70	86

Abbreviations: APRI, aspartate aminotransferase/platelet ratio index; LS, liver stiffness; LSPS, LS × spleen diameter/platelet count; SS, spleen stiffness; US, ultrasonography.

varices.[15] In a cross-sectional evaluation among patients with compensated cirrhosis, a platelet cutoff of 150,000 had a negative predictive value of 96% and a negative predictive value of 15% for medium/large EV.[16] Taken alone, platelet count is not accurate enough to either diagnose or exclude CSPH or gastroesophageal varices in patients with compensated cirrhosis[17]; however, platelet count in combination with other noninvasive parameters improves the predictive value of the noninvasive diagnosis of CSPH.

APRI (aspartate aminotransferase/platelet ratio index) score was introduced by Wai et al in 2003 as a simple noninvasive index for the diagnosis of significant fibrosis and cirrhosis in chronic hepatitis C patients.[18] A specific APRI score cutoff value was correlated for predicting HVPG $\geq$ 12 mm Hg with a diagnostic accuracy of 68%.[19] FibroTest (a noninvasive score designed to diagnose fibrosis, combining α2-macroglobulin, haptoglobin, gamma-glutamyl transterase (GGT), total bilirubin, and apolipoprotein A1) correlated well with HVPG values in a large population of patients with various liver diseases; however, the diagnostic value of FibroTest was not significantly different from platelet count or Child–Pugh score for PH, and no cutoff value with good sensitivity or specificity for CSPH could be established.[20] Platelet count-to-spleen length ratio may exclude the presence of EV in patients with compensated cirrhosis; however, the ratio is not accurate enough to replace endoscopy for identification of high-risk EV.[21]

The ability to evaluate liver stiffness (LS) via transient elastography (TE; FibroScan) has proven to be very accurate in discriminating between patients with and without CSPH. LS correlates strongly with HVPG values up to 12 mm Hg for hepatitis C virus (HCV)-related and alcoholic cirrhosis,[22] albeit the optimal LS cutoff value for CSPH was higher in alcoholic cirrhosis as compared with HCV-related cirrhosis.[23] At a cutoff value of 21 kPa, LS is highly specific for CSPH[24] and had a 100% negative predictive value for the occurrence of PH-related complications.[25] Current recommendations per the 2015 Baveno conference are that LS values of 20 to 25 kPa or more, alone or combined to platelets and spleen size, is sufficient to rule in CSPH in patients with virus-related cirrhosis.[13] LS, independent of other markers of severity of liver disease and synthetic liver function, is able to predict future risk of hepatic decompensation, hepatocellular carcinoma (HCC), and overall mortality in a dose-dependent manner.[26] LS has limitations: several other tissue abnormalities can contribute to increased LS (inflammation, infiltrative diseases, cholestasis) irrespective of fibrosis, and should be considered as possible cofounders of the relationship between LS and portal pressures.[9] Unreliable LS results are also independently associated with obesity (body mass index > 30), meal ingestion, and operator inexperience.[27]

Spleen stiffness (SS) measurement via TE has been investigated as a potential noninvasive surrogate for PH with promising results. In some studies, SS shows a closer correlation with HVPG and CSPH compared with LS.[28,29] SS via TE is limited to 70% of cases and, for technical reasons, is closely dependent on the presence of increased spleen size, therefore limiting the use of SS in routine clinical practice.[9]

Magnetic resonance elastography (MRE) is a relatively new technique that evaluates both LS and SS while overcoming the limitations of US-elastography methods (need for an acoustic window, lack of sensitivity due to body habitus).[9] MRE has been shown to be accurate in the staging of liver fibrosis in nonalcoholic fatty liver disease[30]; however, data regarding the diagnostic performance of MRE for CPSH are very limited.

The combination of different methods which assess different pathophysiological components of PH has been studied to improve upon the diagnostic accuracy of single tests. One novel scoring parameter in particular integrates LS, spleen diameter, and platelet count into a single ratio, LSPS (LS × spleen diameter/platelet count),[31] which had superior performance compared with individual noninvasive measures and allowed for a single cutoff value combining both sensitivity and specificity >80% in identifying patients with CSPH confirmed via HPVG measurement.[32,33]

Esophageal Varices

Given that EV appear at an HVPG of at least 10 mm Hg, patients with EV, by definition, have CSPH. However, CSPH is

present in about 50 to 60% of patients with compensated cirrhosis without EV.[2] Patients with compensated cirrhosis and EV have a worse prognosis than those without EV.[34]

Previous recommendations indicate that all patients with cirrhosis should be screened by esophagogastroduodenoscopy (EGD) for varices at diagnosis.[35] Many studies have looked for noninvasive ways of determining the presence of high-risk varices (varices which require therapy: medium/large varices, varices with red wale marks) in an effort to avoid unnecessary screening endoscopy. Platelet count,[17] platelet count-to-spleen length,[21] LS measurement,[22,36] and SS measurement[28] have shown limited diagnostic accuracy in predicting the presence of EV and are not recommended for diagnosis.

Some noninvasive parameters, however, are accurate enough to rule out high-risk varices in patients with compensated cirrhosis. LSPS values have certain cutoffs which are able to exclude high-risk EV.[31–33] Recent recommendations propose that patients with LS $<$ 20 kPa in conjunction with a platelet count $>$ 150,000 have a very low risk of having high-risk varices ($<$5%) and can therefore avoid screening endoscopy; these patients can be followed up with yearly repetition of platelet count and LS by TE.[13]

In patients with compensated cirrhosis, EV develop at a rate of 7% per year, with progression from small to large varices occurring at a rate of 10 to 12% per year.[37] Current expert opinion suggests that if liver injury is ongoing (active drinking, lack of sustained virologic response (SVR) in HCV) and/or cofactors of disease are present (obesity), surveillance endoscopy for patients without varices during screening EGD should be repeated at 2-year intervals, whereas in the absence of ongoing liver injury, 3-year intervals are sufficient.[13] Likewise, in patients with small varices on screening endoscopy who are not candidates for primary prophylaxis, surveillance endoscopy should be repeated at yearly intervals in patients with ongoing liver injury or present disease cofactors; otherwise, in the absence of ongoing liver injury, these patients may be screened at 2-year intervals.[13]

Primary prophylaxis of VH is indicated for patients with medium/large varices, patients with small varices with "red wale" signs, and decompensated patients with small varices.[38] Treatment consists of either a nonselective β-blocker (NSBB; propranolol, nadolol, or carvedilol), which exert their effects by causing splanchnic vasoconstriction and reducing portal venous inflow, or esophageal variceal ligation (EVL), which consists of placing rubber bands around EV in repeated sessions until they become obliterated. A meta-analysis comparing NSBBs to EVL showed that EVL was associated with lower rates of upper gastrointestinal bleeding, with no difference in mortality.[39] Therefore, current recommendations are for either NSBB or EVL for the prevention of first variceal bleed in patients with medium or large EV, with choice of treatment based on local resources and expertise, patient preference and characteristics, side effects, and contradictions.[13] Advantages of NSBBs include ease of administration, low cost, and no need for expertise; disadvantages are that ~15% of patients may have absolute or relative contraindications to therapy, and another 15% require dose reduction or discontinuation due to common side effects, including fatigue and weakness.[40] Advantages of EVL are that it has few contraindications and does not require medication; disadvantages include a small risk of procedural complications (sedation, strictures, esophageal ulceration) and the necessity of surveillance endoscopies as EV recurrence approaches 90%.[2] In the only randomized control trial comparing the combination of NSBBs plus EVL versus EVL alone in primary prophylaxis of VH, there was no difference in the incidence of bleeding or death between the two groups[41]; therefore, combination therapy is not recommended for primary prophylaxis. Current recommendations for primary prophylaxis for patients with small varices that are high risk (red wale marks or Child–Pugh class C) are treatment with NSBB; further evidence is needed to confirm the benefit of NSBBs in patients with low-risk small varices.[13]

Acute VH is a medical emergency, with a 5-year mortality varying from 20% (as an isolated decompensating event) to up to 80% (when VH presents with ascites or encephalopathy).[42] An HVPG $\geq$ 20 mm Hg (measured within 24 hours of admission) is a strong predictor of early rebleeding and death.[43] Treatment consists of volume resuscitation, vasoactive drugs to reduce portal pressures (octreotide), packed red blood cell transfusions to a target hemoglobin between 7 and 8 g/dL, antibiotic prophylaxis with intravenous ceftriaxone, and urgent endoscopy within 12 hours.[13] If a variceal source is confirmed, EVL should be performed. Despite these efforts, up to 20% of VH episodes can be refractory to standard therapy and persistent bleeding or severe rebleeding is best managed by polytetrafluoroethylene-covered transjugular intrahepatic portosystemic shunt (TIPS).[2] Given the high rebleeding risk (60% in the first year, with a mortality of up to 33%), treatment for patients who have recovered from an episode of acute esophageal VH (secondary prophylaxis) also includes an NSBB in addition to EVL (combination therapy).[2] TIPS is the recommended rescue therapy for patients who experience recurrent hemorrhage despite combination therapy.[13]

Gastric Varices

Gastric varices (GV) occur in ~20% of patients with cirrhosis. GV are commonly classified per Sarin's classifications: GOV type 1 (GOV1) are EV extending below the cardia and into the lesser curvature (75% of GV); GOV type 2 (GOV2) are those extending into the fundus; isolated GV type 1 (IGV1) are located in the fundus; and isolated GV type 2 (IGV2) are located elsewhere in the stomach.[44] Risk factors for GV bleeding include location (IGV1 $>$ GOV2 $>$ GOV1), large size, presence of red spots, and severity of liver dysfunction.[45]

Evidence for primary prophylaxis of GV is scarce. One randomized trial compared endoscopic injection of cyanoacrylate, NSBBs, and observation in patients with large GOV2 and IGV1. Cyanoacrylate injection was associated with lower bleeding rates than NSBBs and observation, but survival in cyanoacrylate injection and that in NSBB were not different, with both higher than observation alone.[46] As NSBBs are the least invasive treatment, current recommendations are for NSBBs in primary prophylaxis of VH from GOV2 or IGV1, whereas guidelines for primary prophylaxis of EV can be assigned to GOV1, given the lack of evidence for GOV1.[13]

Given the lack of evidence for TIPS and balloon-occluded retrograde transvenous obliteration (BRTO) in the primary prophylaxis of GV, neither can be recommended as such.[2]

The initial management of gastric VH is similar to that of esophageal VH (volume resuscitation, blood transfusion, vasoactive drugs, antibiotics, urgent endoscopy). A meta-analysis comparing cyanoacrylate injection versus EVL in endoscopic therapy of gastric VH (primarily GOV1 varices) shows that both therapies are equally effective for initial hemostasis, but cyanoacrylate injection is associated with a significantly lower rebleeding rate.[47] As such, current recommendations for the treatment of actively bleeding GOV1 are for either EVL (if technically feasible) or cyanoacrylate glue injection.[13] TIPS is very effective in the treatment of bleeding GV, with more than 90% success rate for initial hemostasis,[48] and is the preferred treatment for the control of bleeding from GOV2 or IGV1.[2] For secondary prophylaxis, the combination of NSBBs and either EVL or cyanoacrylate injection is first-line therapy to prevent rebleeding, whereas TIPS or BRTO are first-line treatments to prevent rebleeding in patients who have recovered from GOV2 or IGV1 hemorrhage.[2]

Ascites

Ascites refers to the pathological accumulation of fluid within the peritoneal cavity. It is considered a decompensation event with a 1-year mortality rate of 20%.[49] PH tends to be the primary pathophysiological mechanism, with a portal pressure $>$ 12 mm Hg generally necessary for fluid retention.[50] Ascitic fluid analysis and calculation of the serum-ascites albumin gradient ($\geq$1.1 g/dL) accurately diagnose PH-related ascites in 97% of cases.[51] Patients with cirrhosis and PH have a markedly reduced systemic vascular resistance and mean arterial pressure with compensatory increase in cardiac output, therein resulting in a hyperdynamic circulation. As cirrhosis progresses, this process is insufficient, leading to activation of sodium-retaining neurohumoral mechanisms with subsequent water and sodium retention via the renin–angiotensin–aldosterone system, sympathetic nervous system, and antidiuretic hormone release, leading to the development of ascites and often hyponatremia as well.

The mainstays of treatment in patients with cirrhosis and ascites include dietary sodium restriction of less than 2,000 mg/day and oral diuretics (oral spironolactone with or without oral furosemide).[52] Removal of the offending etiology of liver disease, particularly alcohol consumption, can result in dramatic improvement in ascites, resulting in better response to medical therapy or even resolution of ascites.[53]

Refractory ascites, occurring in fewer than 10% of patients with cirrhosis and ascites, is defined as fluid overload that is unresponsive to sodium-restricted diet and high-dose diuretic treatment or recurs rapidly after therapeutic paracentesis.[54] Failure of diuretic therapy may present as either minimal to no weight loss together with inadequate urinary sodium excretion or development of clinically significant complications of diuretics, including encephalopathy, renal failure, hyponatremia, or hyperkalemia.[53] Treatment options for refractory ascites include serial therapeutic paracenteses, TIPS, and liver transplantation. Paracenteses performed up to every 2 weeks can control ascites. Intravenous albumin infusion (25%) of 6 to 8 g per liter of fluid removed following a single large-volume paracentesis of greater than 5 L is commonly used in practice to prevent postparacentesis circulatory syndrome.[53] There is some evidence that polytetrafluoroethylene-covered TIPS improves 1-year transplant-free survival of selected patients with cirrhosis and recurrent ascites as compared with serial paracenteses.[55] Careful patient selection is paramount in achieving a good clinical outcome post-TIPS. Cross-sectional imaging and an echocardiogram should be pursued prior to TIPS. In patients perceived to be high risk (age $>$ 60 years, MELD $>$ 15, bilirubin $>$ 4, Child–Pugh C, PVT), the decision-making process to proceed to TIPS should occur through a multidisciplinary approach.[56]

Hepatic Encephalopathy

HE, or portosystemic encephalopathy, refers to the spectrum of neurocognitive manifestations in patients ranging from altered sleep cycle and decreased attention span, to complete disorientation and comatose state. The risk of the first bout of overt HE is 5 to 25% within the first 5 years after cirrhosis diagnosis, depending on the presence of risk factors.[57] HE is an ominous sign in chronic liver disease and is considered a decompensation event, with median survival in patients with cirrhosis decreasing from $>$8 years to $\sim$2 years.[49] In advanced liver disease, damaged hepatocytes and the development of portosystemic shunts result in ammonia and other nitrogenous compounds being poorly metabolized by the liver as well as bypassing the liver and accumulating in the systemic circulation, where they cross the blood–brain barrier and result in astrocyte swelling.[58] Overt encephalopathy is generally transient and linked with a precipitating event, such as dehydration, the use of sedatives, constipation, renal failure, infection, or gastrointestinal bleeding. Controlling precipitating factors of HE is of paramount importance because nearly 90% of patients can be treated with just correction of the precipitating factor.[59]

Lactulose, a nonabsorbable disaccharide, is considered the first choice for treatment of episodic HE as well as for secondary prophylaxis of HE.[57] Antibiotics have historically been used in the setting of HE, with rifaximin becoming the antibiotic of choice in the treatment of HE due to its safety, efficacy, and tolerability.[58] Combination therapy of rifaximin with lactulose should be considered for recurrent HE on lactulose or severe HE.[60] Many other drugs have been used for the treatment of HE, but data to support their use are limited. However, most of these drugs can safely be used despite their limited proven efficacy: these include intravenous L-ornithine L-aspartate, neomycin, metronidazole, and zinc.[57]

It is estimated that 5 to 35% of patients who undergo TIPS develop new or worsened HE postprocedure, with 3 to 7% of patients developing HE that is refractory to medical therapy, requiring shunt modification or emergent liver transplantation.[61] A major risk factor for HE after TIPS is having recurrent HE prior to undergoing TIPS, which should strongly be considered as a potential contraindication for TIPS.[58] In cases of

refractory HE post-TIPS, shunt revision has been shown in one retrospective study to relieve symptoms of HE 18 to 27 hours postrevision with no recurrence of HE at a mean follow-up of 74 weeks.[62] However, shunt revisions may result in increased PH with subsequent worsening of varices or refractory ascites[63]; thus, careful consideration should be made regarding which patients qualify for TIPS or post-TIPS revision. One study illustrated that neither rifaximin nor lactulose prevented post-TIPS HE any better than placebo.[64] As a result, routine prophylactic therapy (lactulose or rifaximin) is not recommended for the prevention of post-TIPS HE.[57]

Conclusion

The severity of PH clearly translates to an increased mortality risk. The continual assessment of PH and its complications with various methods such as laboratory markers in combination with imaging modalities over the past few years has greatly advanced our ability to predict, treat, and monitor those patients with CSPH with fewer invasive tests. If there is any uncertainty based on noninvasive testing and it is critical to understand the degree of portal hypertension, transjugular portal pressure measurement remains the gold standard to accurately assess portal pressures and can help guide management. As we have more knowledge about the cause and treatment of advanced liver diseases that can lead to CSPH as well as better refinement of treatments for CSPH, we are able to manage our patients with cirrhosis and CSPH better. Patients with CSPH are typically best managed by a multidisciplinary team consisting of gastroenterologists/hepatologists and interventional radiologists to optimally delay the need for liver transplant or more safely bridge patients to liver transplantation.

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